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Environmental Burden of Disease in Europe: Assessing Nine Risk Factors in Six Countries

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Short title: Environmental burden of disease in Europe

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Abstract

Background: Environmental health effects vary considerably with regard to their severity, type of disease, and duration. Integrated measures of population health, such as environmental burden of disease (EBD), are useful for setting priorities in environmental health policies and research. This review is a summary of the full EBoDE project report.

Objectives: The Environmental Burden of Disease in European countries (EBoDE) project was set up to provide assessments for nine environmental risk factors relevant in selected European countries (Belgium, Finland, France, Germany, Italy, and the Netherlands).

Methods: Disability Adjusted Life Years (DALYs) were estimated for benzene, dioxins, second-hand smoke, formaldehyde, lead, traffic noise, ozone, particulate matter ($PM_{2.5}$), and radon, using primarily WHO burden of disease data, (inter)national exposure data, and epidemiological or toxicological risk estimates. Results are presented here without discounting or age-weighting.

Results: About 3-7% of the annual burden of disease in the participating countries is associated with the included environmental risk factors. Airborne particulate matter ($PM_{2.5}$) is the leading risk factor associated with 6,000-10,000 DALYs per year and a million people. Second-hand smoke, traffic noise (including road, rail, and air traffic noise), and radon had overlapping estimate ranges (600 to 1,200 DALYs per million people). Some of the EBD estimates, especially for dioxins and formaldehyde, contain substantial uncertainties that could be only partly quantified. However, overall ranking of the estimates seems relatively robust.

Conclusions: With current methods and data, environmental burden of disease estimates support meaningful policy evaluation and resource allocation, including identification of susceptible groups and targets for efficient exposure reduction. International exposure monitoring standards would enhance data quality and improve comparability.

Background

Scientific evidence shows clearly that environmental risk factors affect human health. Properly targeted and followed-up environmental health policies, such as the coal burning ban in Dublin in 1990 (Clancy et al. 2002) and the smoking ban in public places in Rome in 2005 (Cesaroni et al. 2008) have demonstrated significant population health benefits.

In order to develop effective policy measures and focus research efforts, it is important to prioritize environmental risk factors based on their health impact. Environmental burden of disease (EBD) measures can be used to express diverging health effects in one unit, such as disability adjusted life years (DALYs). DALYs give an indication of the equivalent number of healthy life years lost in a population due to premature mortality and morbidity (Murray and Lopez 1996).

The Dutch National Institute for Public Health and the Environment (RIVM) conducted one of the first systematic European studies using DALYs to compare the health impact of various environmental risk factors (Hollander et al. 1999). The study highlighted that only a few top ranking risk factors produced over 90% of the EBD.

WHO included a ranking of selected environmental exposures in the World Health Report 2002 (WHO 2003) addressing more than a dozen risk factors from a global point of view (Prüss-Üstün et al. 2003) and provided methodological guidance (WHO 2013). OECD compared EBD with monetary impacts in the Environmental Outlook (OECD 2001). More specific EBD studies have looked at e.g. indoor air (De Oliveira Fernandes et al. 2009, Logue et al. 2012,), chemicals (Prüss-Üstün et al. 2011), second-hand smoke (Öberg et al. 2011), and foodborne pathogens (Havelaar et al. 2012). Some of these studies used expert elicitation (De Oliveira Fernandes et al.

2009, Prüss-Üstün and Corvalan 2006), others reviewed results from previous studies (Prüss-Üstün et al. 2011) or used a 'bottom up' data-driven approach to calculate DALYs (Havelaar et al. 2012, Logue et al. 2012).

The current work aimed to test the availability of data and applicability of methods for a data-driven European multinational comparison of the EBD. By looking at the environmental causes of the burden of disease, we provide important information for prioritizing and motivating preventive policies, such as reducing air pollution, traffic noise, and second-hand smoke.

Objectives

The EBoDE project aimed to provide harmonized EBD assessments for the countries participating. Specifically, it aimed to:

- Prioritize selected environmental exposures relevant for the European situation based on their annual health impacts;
- Make data-driven EBD assessments comparable between countries and between environmental risk factors;
- Assess variation and uncertainty in the input parameters and results;
- Assess data availability and method applicability for this type of EBD assessment.

The current paper presents an overview of the results of the EBoDE project. We focus in this paper on the overall results, i.e. comparison of the risk factors. More details about the methodology and data are available in the full EBoDE project report (Hänninen and Knol, 2011).

Methods

The EBoDE –project was launched in 2009 at a WHO meeting (WHO 2009a). Below, methods, data and results are briefly described. See Hänninen and Knol 2011 for more details.

Selection of environmental risk factors, health endpoints and exposure-response functions

Environmental risk factors were selected by the project group based on known public health impacts, high individual risks, public concern, economical interests and pragmatic reasons related to data availability. The nine selected risk factors were benzene, dioxins [including furans and dioxins like polychlorinated biphenyls (PCBs)], second-hand smoke (SHS), formaldehyde, lead, traffic noise (including road, rail and air traffic noise), ozone, airborne particulate matter and radon.

Health endpoints defined in the International Classification of Diseases for each risk factor (Table 1) were selected based on WHO systematic reviews, guidelines and other methods identified in a non-systematic literature review conducted in 2009 as part of the current work (see references in Table 1). Exposure-response functions (ERF) were selected from international recent meta-analyses, WHO guidelines or, if lacking, individual studies published in peer reviewed literature. In some cases, only limited evidence was available; this is especially the case for formaldehyde, which uses a relative risk from a single study.

The EBD was only estimated for exposures above defined thresholds, if any, using a comparative risk assessment method based on a counterfactual exposure distribution that would result in the lowest population risk. The feasibility of reaching the counterfactual exposure levels in practice was not considered.

Estimation of the environmental burden of disease

Three different methods (Methods 1a, 2a, or 2b) were used to estimate the EBD, depending on the type of ERF estimate available for each exposure-outcome pair [either a relative risk (RR) based on environmental epidemiology, or a unit risk (UR) based on toxicological or occupational data], and on the availability of a WHO baseline burden of disease (BD) estimate (WHO 2009b) for the outcome. The method used for each exposure-outcome relation is listed in Table 1.

When a WHO BD was available for a given outcome, the environmental burden of disease (EBD) was estimated based on the population attributable fraction (PAF) for that outcome in relation to each exposure of interest,

$$EBD = PAF \times BD. \quad [1]$$

Two methods were used to estimate the PAF, depending on the type of ERF estimate available: *Method 1a.* For exposure-outcome pairs with an RR estimate, the PAF is derived as (Rockhill et al. 1998):

$$PAF = [p \times (RR\text{-}1)] / [p \times (RR\text{-}1) + 1], \quad [2]$$

where p is the proportion of population exposed and RR is the relative risk at the level of exposure.

Method 2a. Unit risks (UR) were used to estimate the PAF for exposure-outcome pairs without RR estimates available. UR, which are an estimate of the number of cases expected at a certain level of exposure, allow for direct estimation of the number of attributable cases (AC) from the exposure data:

$$AC = E \times UR \times P, \quad [3]$$

where E is the exposure level, UR is the unit risk, and P is the size of the exposed population. The PAF is estimated from the AC as:

$$PAF = AC / I, \quad [4]$$

where I is the total incidence of the studied endpoint. The *EBD* is then estimated using equation 1. This method will slightly overestimate the impact of the environmental exposure on mortality by including also non-fatal cases in AC, but allows for using standard WHO burden of disease data. The overestimation depends on the site of the cancer in question and is small for highly fatal cancers (e.g. lung cancer) but larger for less fatal cancers (like childhood leukemia) and total cancers.

Method 2b. For outcomes without a WHO BD estimate available (e.g., severe sleep disturbance), the EBD was estimated as

$$EBD = AC \times DW \times L, \quad [5]$$

where AC is the number of attributable cases (estimated using UR and Equation 3), DW is the disability weight characterizing the severity of the disease [ranging from 0 (perfect health) to 1 (death)] and L is the average number of years lived with disability (YLD) for morbidity effects, or years of life lost for mortality (YLL).

Results were calculated both using the WHO Global Burden of Disease 2004 approach with age weighting and discounting (3%) and without age-weighing and discounting (as done in the Glogal Burden of Disease 2010 –study). Additionally, as some of the health outcomes such as cancers have long incubation periods between exposure and clinical detection of the disease,

these lag-times were considered in the discounted model. However, in this paper all results are presented without discounting and age-weighting. Discounting affects significantly the magnitude of the estimates in case of premature mortality and chronic conditions, up to a factor of two. However, comparisons of the discounted and non-discounted results showed that the ranking of exposures was not very sensitive to the choice of discounting and age-weighting or not. The reader is referred to the project report (Hänninen and Knol, 2011) for a more comprehensive discussion on this.

Selection of health endpoints

Health endpoints and dose-response coefficients are summarized in Table 1.

Benzene effects were estimated for leukaemia, including morbidity and mortality. Other proposed health endpoints were not included, because occurring only at high exposure levels, typical of occupational settings. We used the exposure response function as recommended by the WHO Air Quality Guidelines (WHO 2000).

The effect of exposure to dioxins and dioxin-like PCBs were estimated on cancer (all cancer types). The non-cancer effects were not considered due to difficulties in estimating the exposure-response relationships and the other input parameters necessary for estimating DALYs and therefore the estimates were calculated by first assuming all attributable cancer cases fatal during the first year after clinical detection and then using PAF from Eq 4 in Method 2a. Leino et al. (2008) assumed a linear exposure-response relationship for excess cancers associated with dioxin intake. They estimated the health risk for toxicity equivalent intake assuming additivity of the toxicity of the different types of dioxins and all cancer cases to be lethal.

The EBoDE calculations use the Leino et al. (2008) approach, but the results have been corrected with an updated cancer slope factor 1×10-3 per pg/kg/d of dioxin intake of the U.S. Environmental Protection Agency (NAS 2004; USEPA 2003). The assumption that all cancers are lethal may lead to overestimation of the impacts.

Out of the large number of health endpoints that SHS is associated with, we selected mortality and morbidity due to lung cancer and ischemic heart disease (IHD), morbidity due to onset of asthma (both in children and in adults), lower respiratory infections and acute otitis media. For the other health endpoints mentioned above, strong evidence is available, but the necessary disease statistics were lacking. For the SHS-related burden of disease calculations, we have followed the recent WHO methods on the global estimation of disease burden from SHS (Öberg et al. 2011). The selected outcomes are being applied only to non-smokers, i.e. to the non-smoking disease burden. To that effect, the disease burden due to active smoking has been deduced from the total disease burden, by country (based on total disease burden and active smoking disease burden by country provided by WHO; update 2002 based on Ezzati et al. (2004)).

The development of asthma in toddlers was the only health endpoint included for formaldehyde (Rumchev et al. 2002). Sinonasal cancer, observed at occupational exposure levels, has been ruled out by WHO Air Quality Guidelines working groups concluding that there is no epidemiological or toxicological evidence that formaldehyde would be associated with sinonasal cancer at levels below 1 mg/m^3 (WHO 2000, 2010a). The WHO Guidelines for Indoor Air Quality (WHO 2011) use eye irritation as the main health end-point associated with formaldehyde; however, due to difficulties in estimating a burden of disease from irritation this endpoint was not included in our calculations.

The estimates for lead include two endpoints that have been shown to be relevant at current exposure levels: mild mental retardation (due to IQ loss) and hypertensive disease (due to rise in systolic blood pressure). These associations exist at levels below 100 µg/liter (Canfield et al. 2004; Carta et al. 2005; Walkowiak et al. 1998). Therefore, an extrapolation of the exposure-response curve to the range below 100 µg/l seems adequate. Lanphear et al. (2005) proposed a log-linear model for this curve.

Health end-points associated with traffic noise included high sleep disturbance and Ischemic Heart Disease (IHD) (Babisch 2006, 2008; Miedema and Vos 2007). Hypertension and related heart disease due to aircraft noise was not considered because no clear review could be identified at the time. Nevertheless, since causal relationships are very likely and have been reported recently, this health effect may be considered in the future (Babisch and Kamp 2009). For railway noise no significant associations with hypertension and IHD could be identified either (Barregard et al. 2009). Effects on cognition and severe annoyance were excluded, as these are difficult to quantify.

For ozone, as well as for PM, we followed the quantification approach as laid out in the Clean Air For Europe (CAFE) project and based on WHO European Centre for Environment and Health and CLTRAP Task Force on Health consultations (Hurley et al. 2005). Health effects that are taken into consideration include total non-violent mortality, minor restricted activity days (MRADs), and cough and lower respiratory symptoms (LRS) in children aged 5-14 years (WHO 2008).

$PM_{2.5}$ and PM_{10} both serve as indicators of a complex mixture of physically and chemically heterogeneous composition. The burden of disease related both to PM_{10} and to $PM_{2.5}$ exposures

were calculated, but due to the overlap between these two indicators, in the aggregate results only the results for $PM_{2.5}$ are presented. For $PM_{2.5}$, we calculated the burden of disease for cardiopulmonary mortality, lung cancer mortality, total non-violent mortality, chronic bronchitis and restricted activity days (RAD; defined by Hurley et al., 2005). Due to the overlap between the different mortality endpoints, we report only cause specific mortality in the aggregate results. For mortality, we used the relative risks as provided by Pope (Pope et al. 2002; WHO 2006ab). For morbidity, relative risks are based on the thorough review made for the CAFE estimates by Hurley et al. (2005) and WHO (2006b).

Radon effects are usually presented as additional cases of lung cancer at a certain exposure (i.e. unit risk model). In order to account for the interaction with smoking, however, a relative risk model seems more appropriate. We therefore calculated results using both a unit risk model and the relative risk model (Methods 1a and 2a). The RR method (1a) results are presented as the final results. The relative risk model, as suggested by the meta-analysis of Darby et al. (2005), assumes the lung cancer risk from radon to be linearly proportional to the radon exposure, but also to the background lung cancer rate caused by tobacco smoking and, to a lesser extent, by exposure to second-hand smoke, ambient air particulate matter and possibly some occupational exposures.

Exposure data

Calculations were carried out for the year 2004, the latest year for which exposure and health data were sufficiently available for the studied countries. Exposure data were preferably collected from internationally harmonized sources (Table 2), but in the case of benzene, dioxins, formaldehyde, and lead, (complementary) national data were needed. Population average data

were used for all age groups when age-group-specific data were lacking. More details are available in the project report (Hänninen and Knol 2011, Chapter 3).

Uncertainty estimation and alternative analyses

Many factors can contribute to uncertainty in EBD estimates (Knol et al. 2009), including the selection of risk factors and health effects, exposure data, exposure-response functions, and methodological choices. Some of these sources of uncertainty can be handled quantitatively, whereas others can only be described qualitatively. For the quantitative part, we have estimated statistical confidence intervals based on the uncertainty ranges of the exposure-response functions. In addition, we carried out several alternative analyses to explore the robustness and sensitivity of our results. We tested the effect of lag-times from exposure to the onset of the disease and compared PM and ozone results to those obtained by using life tables, and used a variety of different assumptions for our input data and models in selected scenarios. Details of these analyses are available in the project report (Hänninen and Knol 2011 Chapter 5).

For the qualitative part, we used expert judgment (provided by the thematic experts participating in the project) to evaluate the knowledge base to support the claim of causality between exposure and effect and other main factors affecting the model uncertainty.

Results

Unless otherwise specified, all DALYS are presented as population weighted non-discounted and non-age-weighted annual averages. European results are calculated as weighted averages accounting for the size of population in each participating country.

Overall results

The central EBD estimates per environmental risk factor ranged from 2 to 10,000 DALYs per million people in the six participating countries (Belgium, Finland, France, Germany, Italy, and the Netherlands). The relative population-weighted contributions of the risk factors are shown in Figure , dominated by particulate matter (68%), followed by second-hand smoke and traffic noise (8% each) and radon (7%). The estimated EBD was clearly dominated by $PM_{2.5}$, which accounted for about 4,500-10,000 DALYs per million people, followed by SHS (600-1,200), radon (450-1,100) and traffic noise (400-1,500) (Figure 2). Estimates for lead (100-900), ozone (30-140) and dioxins (200-600) were classified to have medium public health impacts. Benzene (2-4) and formaldehyde (< 2) had relatively the lowest public health impacts. Ranking orders varied between countries. Figure 2 shows the estimated EBD and the quantitative ranges of the estimates between the six participating countries. More elaborate expert judgment of overall uncertainties is presented in the full report (Hänninen and Knol 2011), where the statistical uncertainty of the exposure-response functions are combined with the estimated level of certainty of the underlying knowledge on causality.

For six risk factors the public health impacts are dominated either by morbidity (formaldehyde, lead and traffic noise) or by mortality (benzene, dioxins, and radon). The selection of health endpoints may be partly responsible for this finding. In total, the selected risk factors are associated with 1.6 million years of life lost in the participating countries, or 6,900 YLL per million inhabitants.

Health endpoint specific estimates ranged from 0.1 to 4,600 DALYs per million people, with the highest impacts for cardiopulmonary mortality, lung cancer mortality, and chronic obstructive pulmonary disease (COPD) related to $PM_{2.5}$ exposure (4,600, 1,500 and 1,200 DALYs per

million people, respectively). These were followed by lung cancer (radon; 830 DALYs per million), severe sleep disturbance (traffic noise; 720 DALYs per million) and ischemic heart disease (SHS; 680 DALYs per million).

The total national BoD was estimated to range from 112,000 in Italy to 132,000 DALYs per million people in Finland (WHO 2009b). The nine investigated risk factors contributed 3.3 – 6.9% to the total estimated BoD, with the highest contribution in Italy and the lowest in Finland. In the intermediate countries the contribution of EBD to the total BoD was 6.3% in Belgium, 4.4% in France, 5.4% in Germany and 5.6% in the Netherlands. The risk factor specific DALYs per country are presented in Hänninen and Knol 2011 (pp. 87-93) and in the Supplemental material.

Results and uncertainties by risk factor

Particulate matter

Particulate matter ($PM_{2.5}$) accounted for 68 % of the total estimated EBD, making it the most significant environmental risk factor in our analysis (Figure 1). This is in line with results of similar assessments (de Hollander et al. 1999, Logue et al. 2012, Prüss-Üstün et al. 2011). In the six participating countries $PM_{2.5}$ is estimated to cause 1.8 million DALYs annually and 1.3 million lost life years (i.e. premature mortality only). Overall, 73% of the health impacts due to $PM_{2.5}$ exposure were estimated to be attributed to mortality. The estimated $PM_{2.5}$ impact ranged from 4,600 in Finland and France to 10,500 DALYs per million people in Belgium.

Main uncertainties relate to the exposure-response functions and the potential of double-counting of morbidity effects by combining restricted activity days and lower respiratory symptom days. Overall, PM is the most thoroughly reviewed risk factor included in this study.

Second-hand smoke (SHS)

The EBD related to SHS was estimated to account for 600-1,200 DALYs per million people. This is well in line with a large recent EBD assessment (Öberg et al. 2011) that estimated about 610 DALYs per million people in the Western Europe.

Main uncertainties in our estimates relate to the difference between survey-based exposure measurements, matching between measured exposures and relative risks, and the various assumptions made in applying the method (e.g. assuming that active smokers are not susceptible to SHS). Nonetheless, most evidence for SHS-related impacts is fairly consistent, and estimates of the EBD are considered relatively stable.

Estimated EBD from SHS is remarkably low in France (550 non-discounted DALY/million) and high in Germany (1200), where exposure levels and baseline prevalence of the relevant diseases are higher.

Radon

Exposure to radon was estimated to cause 450 – 1,100 DALYs per million people. The radon-related EBD is the highest in France (1100 non-discounted DALY/million) and Belgium (1100), and lowest in the Netherlands (450). These differences are mainly caused by differences in geologically driven uranium concentrations in the soil, use of different building materials and differences in national mitigation measures.

Traffic noise

Since so many people are exposed to traffic noise (including road, rail and air traffic), the total estimated EBD associated with this exposure is substantial (400 – 1,500 DALYs per million people), despite the relatively small disability weights for severe sleep disturbance (0.07).

DALYs range from 370 per million people in less densely populated Finland up to 1480 DALYs per million people in France. The exposure data, which were derived from the Environmental Noise Directive (2002) reporting from 2007, cover only agglomerations with more than 250 000 inhabitants and roads outside these agglomerations with more than 6 million vehicles per year, railroads with more than 60 000 passages per year and airports with more than 50 0000 flights per year. Therefore, the results are probably an underestimation of the total burden in a country. In addition, only exposure levels above Lnight 50dB (Lden 55 dB) were available, so health impacts could not be estimated for lower exposure levels.

Dioxins, furans and dioxin-like PCBs

The EBD related to dioxins in food was estimated to range from 240 to 580 DALYs per million people. Uncertainties are large: effects of dioxins cannot easily be distinguished from other chemicals; low-dose effects are difficult to assess; thresholds for effects are mostly unknown. Our estimates are based on simplification of assuming each cancer case fatal during the first year when calculating the population attributable fraction (PAF) using Method 2a. Non-cancer effects were not considered due to a lack of dose-response-functions or quantifiable health endpoints. The PAF estimation method used could lead to a slight overestimation of dioxin effects due to counting non-fatal cases in the body count. On the other hand, ignoring non-cancer effects could lead to an underestimation. We were not able to quantify these counteracting uncertainties. The EBD of dioxin exposure varies due to differences in diets and food contamination, and the different methods used to evaluate daily intake.

Lead

Lead was estimated to contribute to 100-900 DALYs per million people. The underlying exposure data had limited population representativeness and were partly based on older data supplemented with trend estimations. Other uncertainties relate to unavailability of exposure-response functions over the complete exposure spectrum, and the aggregation of effects. Lead exposures were the highest in Italy. One of the reasons for this may that the exposures were measured in adults only. In the Netherlands, in contrast, the sample included children aged 1-6 years. Since lead accumulates in the body over the years, this is probably the most important reason for lead-related EBD being relatively low in the Netherlands (220 non-discounted DALY/million) and relatively high in Italy (950). More consistent human biomonitoring data are needed for lead.

Ozone

The acute impacts of tropospheric ozone on public health ranged from 30 to 140 DALYs per million people. Uncertainties in the calculations relate, amongst other issues, to the estimated years of life lost due to mortality and chronic effects. Estimated ozone impacts were highest in the Mediterranean countries, represented here by Italy (140 non-discounted DALY/million). Levels in the Netherlands were the lowest (34), probably because of meteorological factors and relatively high levels of nitrogen oxide.

Benzene

The EBD of benzene in air was estimated to be less than 5 DALYs per million people. Representativeness and comparability of exposure data was estimated to be the largest source of uncertainty.

Formaldehyde

The EBD related to formaldehyde in air was estimated as less than 2 DALYs per million people. Formaldehyde levels in Finland are higher than in many other developed countries due to the types of construction materials used and the relatively tightly sealed buildings.

Main uncertainties related to the difficulties in selection of endpoints, thresholds, and very limited epidemiological data at prevailing exposure levels. We applied a threshold of 100 $\mu g/m^3$ (WHO 2000, 2010a), which is exceeded very rarely in Europe.

Discussion

Policy relevance

Environmental burden of disease (EBD) estimates are aimed to support efficient policy development and resource allocation. International comparisons over a range of environmental risk factors, as presented in this study, form a valuable basis for prioritizing among environmental policies and for international benchmarking. International comparisons can also be a strong incentive for national policy development. Preliminary results of this study were greatly appreciated when presented at the fifth Ministerial Conference on Environment and Health in Parma in 2010 (WHO 2010b). Based on our results, PM is an obvious candidate that requires further reduction, whereas dioxins and formaldehyde seem to be less relevant from a population-wide EBD perspective. However, for these risk factors, policy action also may be required, e.g. for specific susceptible groups. Our approach does not allow for estimating health impacts in specific population groups, e.g. highly exposed (e.g. occupational exposures) or other susceptible groups (gender, age, genetic predisposition). Such information is needed when developing specific policy measures and considering environmental equity, feasibility of policy

measures, developing accountability studies, and evaluating health benefits, wellbeing, risk perception, and associated uncertainties.

Interpretation of the presented EBD estimates in the context of risk management and policy development requires care. Besides the inherent uncertainties, the EBD as calculated here cannot be directly interpreted as the total reduction potential. Some health impacts may always remain because of background concentrations from natural sources and practical limitations in removing anthropogenic pollution. Using expert judgment, Prüss-Üstün et al. (2006) estimated the EBD related to modifiable environmental factors, which may be more relevant from a policy effectiveness perspective. As future research, it would be interesting to investigate the actual use and effect of EBD studies on national or international agenda setting, policy development and policy evaluation.

Uncertainties and limitations

Due to the large amount of data and knowledge needed for EBD calculations, many sources of uncertainties affect the results (Knol et al. 2009). Besides the parametric uncertainties, for which we have calculated numerical uncertainty ranges, we carried out a number of quantitative sensitivity analyses for model uncertainties, and also used expert judgments to provide a qualitative estimate of the knowledge base underlying the claims for causality.

Overall, we believe that the six country averages are likely to provide reasonable estimates of the magnitude of the environmental burden of disease in Western Europe, and that uncertainties will not affect the rank ordering of the estimated impacts of the risk factors, though estimated impacts of SHS, radon, and traffic noise do overlap. However, generalizability to other countries is limited by risk factor-specific issues.

For example, radon exposures are highly variable, and the differences in exposure levels cannot be generalized.

The numerical uncertainty ranges presented here, based solely on uncertainty in the exposure-response functions.,The evaluation of the knowledge base on causality, based on expert judgment, was considered to have the highest reliability for $PM_{2.5}$, SHS, radon and benzene. Medium uncertainties were identified for traffic noise, lead, and ozone, while dioxins and formaldehyde were considered most uncertain. Non-conclusive sensitivity analyses suggest that our overall ranking of risk factors is relatively robust against identified main sources of model uncertainties. Baseline comparison with other data-driven EBD studies (including de Hollander et al. 1999, Logue et al. 2012, OECD 2001) confirms relative robustness of the overall ranking and order of magnitude of the estimates, despite methodological differences and variation in baseline assumptions.

Only impacts for which sufficient evidence and quantitative data were available were included in our EBD estimates. The availability of data and evidence was evaluated by the experts that participated in the study. Health effects that are suspected but not sufficiently researched or monitored, as well as health effects that fall outside the scope of the International Classification of Diseases (ICD) coding system, were not included. Expert elicitation, as used by e.g. Prüss-Üstün et al. (2006) and structured by Knol et al. (2010), may be useful to fill in some of these gaps.

The exposure data we used had varying degrees of temporal, population, and geographical coverage. Exposure data collected with standardized methods over all the participating countries were available for $PM_{2.5}$ and ozone from the European air quality monitoring system (AirBase)

(see Table 2). Radon, SHS, benzene and dioxins had reasonably comparable data. Radon exposures are monitored by national programs and have been extensively reviewed by international research groups (Darby et al. 2005). Second hand smoke exposure questionnaire was conducted in all European countries (EC 2009). Also dioxins have been extensively reviewed, even though in the sampled media there were differences in data availability between the countries. Traffic noise data collection is well defined in the European Noise Directive, but the comparability of the data available from the first phase of this directive had not yet reached these standards at the time of collecting the current data. Lowest exposure data comparability was found for lead and formaldehyde data, for which the assessments were based only on studies with no international standardization in population sampling, seasonal variability and temporal trend estimation. This can be considered surprising. Lead has been a very important pollutant in the past, and policy evaluation and follow-up would require comparable and representative exposure data. In several countries lead exposure levels have been in strong decrease over the last years, as documented for instance for Italy (Alimonti et al. 2011).

International monitoring standards and procedures could strengthen data quality and improve comparability. The current lack of harmonized environmental exposure data is one of the things that hinders comparable EBD assessments and policy evaluation.

Discounting, age weighting and lag-times

When calculating DALYs, it is optional to discount or age-weight the results. Discounting is based on the assumption that future years of healthy life are considered less valuable than years of healthy life at the present time. Non-uniform age-weighting means that a year lived at younger or older ages is given a lower value than a year lived by a young adult. The use of both discount rates and age-weighting has been debated (Anand and Hanson 1997; Arnesen and Nord 1999;

Schneider 2001). Discounting leads to lower valuation of impacts that occur later or last longer, in comparison with immediate effects. This is not favorable for children and future generations, and it devalues preventive measures. The use of age weights is also controversial, as it values the lives of children and elderly less than other lives. Therefore, in this study we have chosen not to discount or age-weigh our main results. The recent Global Burden of Disease 2010 study, coordinated by Institute of Health Metrics (IHME) also rejected discounting and age-weighting (Lim et al. 2012).

We performed additional analyses to explore the effects of discounting and age-weighting (see Hänninen and Knol 2011, p.70). The overall ranking of the risk factors was more or less stable against the alternative discounting procedures. However, the absolute magnitude of the estimated impacts was reduced to one third of the non-discounted value by discounting and age-weighting for diseases associated with substantial premature mortality and chronic diseases, e.g. in case of lung cancer associated with second hand smoke, $PM_{2.5}$ and radon. In other contexts, such as debates over nuclear energy, the health of future generations is often given priority over benefits of the current economy. Moreover, children's health has been set as a priority in the European Environmental Health Action Plan (WHO 2010b). This contrasts with the consequences of discounting and age-weighting, which downscale health impacts in children.

Conclusions

EBD was estimated for nine environmental risk factors (benzene, dioxins, formaldehyde, SHS, lead, traffic noise, $PM_{2.5}$, ozone and radon) in six countries. The highest overall public health impact was estimated for ambient fine particles ($PM_{2.5}$; annually 4,500-10,000 non-discounted DALYs per million in the six participating countries) followed by second-hand smoke (600-

1,200), traffic noise (400-1,500), and radon (450-1,100). Medium impacts were estimated for lead, dioxins and ozone. Lowest impacts were estimated for benzene and formaldehyde. The relative ranking of the risk factors was relatively robust under the uncertainties examined.

EBD assessment is useful for setting research and risk management priorities from the point of view of public health benefits and resource allocation. This may include both the identification of susceptible population groups, and health-based evaluation of the efficiency of potential benefits from exposure reduction policies. Further development of methods to address additional risks and health outcomes would allow a more complete account of health impacts caused by environmental risks. International exposure monitoring standards and activities would improve data availability, strengthen data quality and improve comparability.

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Table 1. Summary of health endpoints, exposure units, exposure/response-relationships and calculation methods.

Risk factor	Selected health endpoints	Population	Exposure estimate	Unit of exposure	Type of ERF	Point estimate of ERF (95% CI) [a]	Reference(s) for ERF	Threshold [b]	Calculation method [c]
Benzene	Leukaemia	All	Annual mean exposure	µg m^{-3}	UR	6.00×10^{-6} (2.20×10^{-6}-7.80×10^{-6})	WHO 2000	0	2a
Dioxin	Total cancer incidence	All	Daily intake of adults	pg/kg/d	UR	1.00×10^{-3} (5.70×10^{-4}-5.10×10^{-3})	Leino et al. 2008; NAS 2004	0	2a
SHS	Tracheas, bronchus and lung cancers [d]	Adult non-smokers	% of exposed	yes/no	RR	1.21 (1.13-1.30)	US S.G. 2006	0	1a
SHS	Ischemic heart disease	Adult non-smokers	% of exposed	yes/no	RR	1.27 (1.19-1.36)	US S.G. 2006	0	1a
SHS	Asthma induction	Adult non-smokers	% of exposed	yes/no	RR	1.97 (1.19-3.25)	Jaakkola et al. 2003	0	1a
SHS	Asthma induction	Children (< 14 yr)	% of exposed	parental y/n	RR	1.32 (1.24-1.41)	Cal-EPA 2005	0	1a
SHS	Lower respiratory infections	Infants (< 2 yr)	% of exposed	parental y/n	RR	1.55 (1.42-1.69)	US S.G. 2006	0	1a
SHS	Otitis media	Toddlers (< 3yr)	% of exposed	parental y/n	RR	1.38 (1.21-1.56)	Cal-EPA 2005; Etzel et al. 1992	0	1a
Formal-dehyde	Asthma aggravation (children) (morbidity only)	Toddlers (< 3 yr)	Annual mean residential indoor concentration	µg m^{-3}	RR	1.017 (1.004-1.025)	Rumchev et al. 2002	100	1a
Lead	IQ loss	Children (< 5 yr)	Blood lead levels	µg/l	UR	0.051 (0.032-0.07)	Lanphear et al. 2005	24	n/a
Lead	Mild mental retardation (morbidity only)	Children (< 5 yr)	Blood lead levels	µg/l	DS [e]	function	-	24	2b
Lead	Hypertensive diseases (morbidity only)	Adults/All	Blood lead levels	µg/l	DS [e]	function	-	50	2b
Lead	Increased blood pressure	Adults/All	Blood lead levels	µg/l	UR	2.50×10^{-2} (1.70×10^{-2}-3.20×10^{-2})	Fewtrell et al. 2003, Schwartz 1995	50	n/a

Risk factor	Selected health endpoints	Population	Exposure estimate	Unit of exposure	Type of ERF	Point estimate of ERF (95% CI) [a]	Reference(s) for ERF	Threshold [b]	Calculation method [c]
Road traffic noise	Severe sleep disturbance (HSD) (morbidity only)	All	Exposure categories	Lnight (dB)	UR	function	Miedema and Vos 2007, WHO 2009c	35	2b
Road traffic noise	Ischemic heart disease (IHD) (mortality and morbidity)	All	Exposure categories	Lday16h (dB)	OR	function	Babisch 2006, 2008	55	1a
Railway traffic noise	Severe sleep disturbance (HSD) (morbidity only)	All	Exposure categories	Lnight (dB)	UR	function	Miedema and Vos 2007, WHO 2009c	35	2b
Aircraft noise	Severe sleep disturbance (HSD) (morbidity only)	All	Exposure categories	Lnight (dB)	UR	function	Miedema and Vos 2007; WHO 2009c	35	2b
Ozone	Total mortality (non-violent)	Adults (> 30 yr)	Ambient SOMO35 level	$\mu g\ m^{-3}$	RR	1.0003 (1.0001-1.0004)	WHO 2006a	70	1a
Ozone	Minor restricted activity days (morbidity only)	Working age (18-64 yr)	Ambient SOMO35 level	$\mu g\ m^{-3}$	UR	0.0115 (0.0044-0.02)	Hurley et al. 2005; WHO 2006b	70	2b
Ozone	Cough days, children (morbidity only)	School children (5-14)	Ambient SOMO35 level	$\mu g\ m^{-3}$	UR	0.093 (0.019-0.22)	Hurley et al. 2005; WHO 2006b	70	2b
Ozone	LRS days in children (excl cough) (morbidity only)	School children (5-14)	Ambient SOMO35 level	$\mu g\ m^{-3}$	UR	0.016 (-0.043-0.08)	Hurley et al. 2005; WHO 2006b	70	2b
$PM_{2.5}$	Cardiopulmonary disease (mortality and morbidity)	Adults (> 30 yr)	Population weighted ambient level	$\mu g\ m^{-3}$	RR	1.0077 (1.0020-1.0132)	Pope et al. 2002; WHO 2006a	0	1a
$PM_{2.5}$	Lung cancer (mortality and morbidity)	Adults (> 30 yr)	Population weighted ambient level	$\mu g\ m^{-3}$	RR	1.012 (1.004-1.020)	Pope et al. 2002; WHO 2006a	0	1a

Risk factor	Selected health endpoints	Population	Exposure estimate	Unit of exposure	Type of ERF	Point estimate of ERF (95% CI) [a]	Reference(s) for ERF	Threshold [b]	Calculation method [c]
$PM_{2.5}$	Chronic bronchitis (new cases) (mortality and morbidity)	Adults (> 27 yr)	Population weighted ambient level	$\mu g\ m^{-3}$	UR	5.33×10^{-5} (1.70×10^{-6}-1.13×10^{-4})	Hurley et al. 2005; WHO 2006b	0	2b
$PM_{2.5}$	Restricted activity days (RAD) (morbidity only)	15-64 yr	Population weighted ambient level	$\mu g\ m^{-3}$	UR	0.0902 (0.0792-0.101)	Hurley et al. 2005; WHO 2006b	0	2b
Radon	Lung cancer (mortality and morbidity)	All	Residential mean level	$Bq\ m^{-3}$	RR	1.0016 (1.0005-1.0031)	Darby et al. 2005, 2006	0	1a

[a]These exposure-response functions are all expressed per 1 unit of exposure. [b]Above which the health impacts are included in the estimates. [c]Different types of calculation methods were applied as described in the Methods section. Method 1a (RR+PAF): Deriving the PAF from epidemiological data; applying the PAF to WHO total burden of disease data. Method 2a (UR+PAF): Estimating PAF from the Unit Risk and total incidence; applying the PAF to WHO total burden of disease data. Method 2b (UR+L+DW): Using a Unit Risk to calculate Attributable Cases (AC); calculating the Burden of Disease: AC x L x DW. [d]The RR for spousal smoking is used as a proxy for any regular exposure (including at work). [e]For lead, a shift in exposure distributions is linked to a unit risk approach.

Function: No point estimate can be given, as the exposure-response function is given by a more complex function.

AI=Attributable Incidence; ARI = Acute respiratory infections; Bq = Becquerel; ERF = Exposure-response function; LCL/UCL lower/upper confidence limit (95%); IHD = Ischemic heart disease; Lday16h = noise level for day and evening; LRS = Lower respiratory Symptoms; PCB = Polychlorinated biphenyls, PAF=Population Attributable Fraction; PM = Particulate Matter; RAD = Restricted activity days; SHS = Second-Hand Smoke, SOMO35 = sum of maximum 8-hour ozone levels over 35 plead (70 $\mu g/m^3$); UR = Unit Risk; RR = Relative Risk; yr = year.

This table is adopted from the full report (Hänninen and Knol, 2011) with the permission of the copyright holder.

Table 2. Sources for exposure data (for more details see Hänninen and Knol 2011, page numbers referred below).

Stressor	Year(s) of original exposure data	Assumptions for trends estimation to 2004	Exposure data sources
Benzene	2004	National trend estimates when applicable	AirBase (2009) data for outdoor levels in 2004; national studies for indoors [a]
Dioxins	1997-2006	No trend assumed	National data for intake [a]
Second-hand smoke	2008	Available data fitted with power functions for trends	National [a] and international survey data for exposures between 1990 and 2008 used for modelling 2004 data; EC, 2009
Formaldehyde	1990-2005	No trend assumed	National indoor concentration data [a]
Lead	1990-2005	National trend estimates	National blood lead level data [a]
Traffic noise	2007[b]	No trend assumed	EC Environmental Noise Directive (END) data
Ozone	2005	No trend assumed	ECT/ACC spatial model based on AirBase (2009) observations and air quality maps
Particulate matter	2005	No trend assumed	
Radon	up-to 2005	No trend assumed	RadonMapping project (http://radonmapping.jrc.ec.europa.eu) and the UNSCEAR 2000 Report

[a]References to sources of national exposure data are presented in the supplemental materials. [b]Target year of END data was set as 2007. The actual collected data contains subsets of data from various years.

Figure Legends

Figure 1. Relative contributions of the nine targeted risk factors to the estimated burden of disease attributed to these risk factors, averaged over the six participating countries. The figure is adopted from Hänninen and Knol, 2011 with permission from the copyright holders.

Figure 2. Ranges for the estimated contributions of the selected environmental risk factors to the burden of disease (DALYs per million people) as population-weighted averages over the six participating countries. The figure is adopted from Hänninen and Knol, 2011 with permission from the copyright holders.

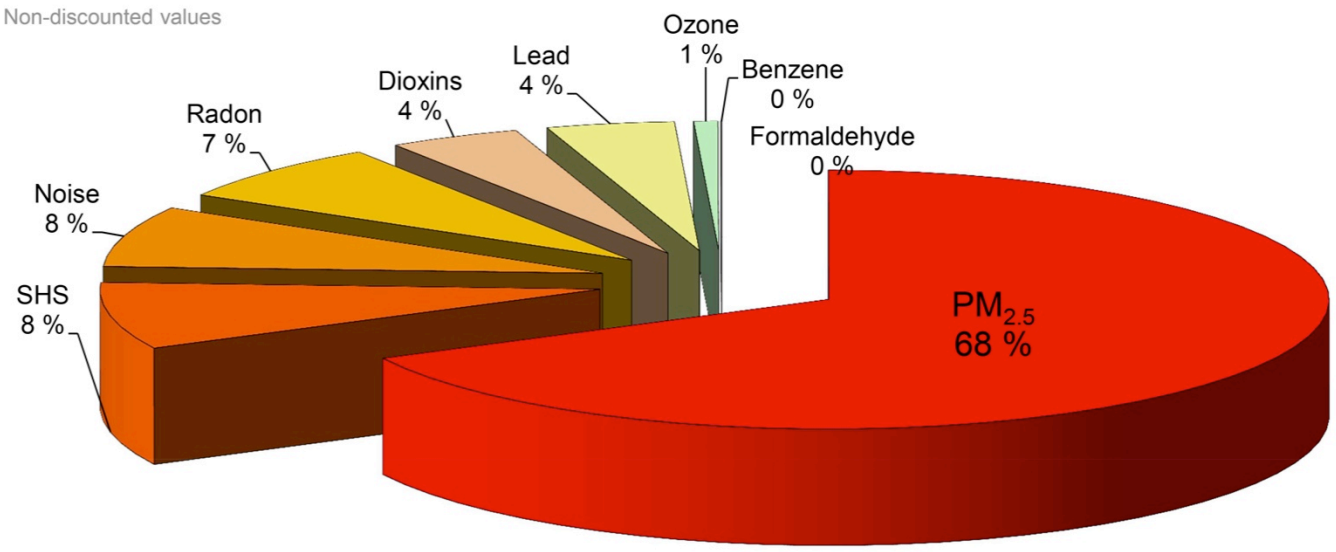
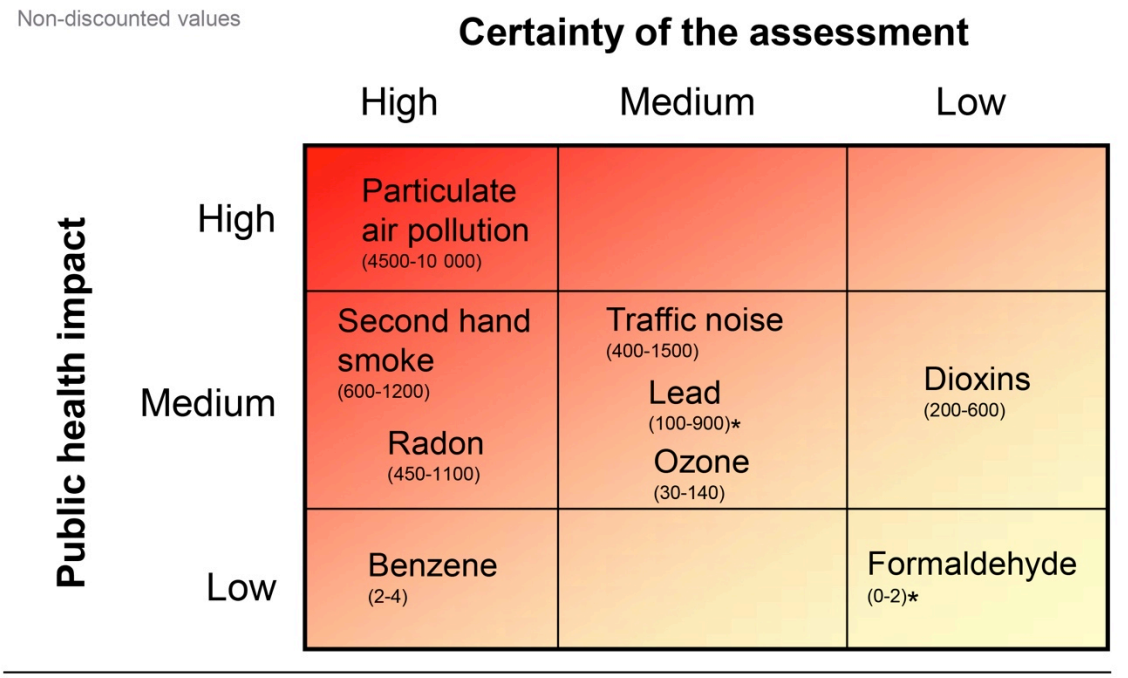

Non-discounted values
Ozone
1 %
Benzene
0 %
Formaldehyde
0 %
Lead
4 %
Dioxins
4 %
Radon
7 %
Noise
8 %
SHS
8 %
PM2.5
68 %

Figure 1.



Numerical values indicate non-discounted DALYs per million people in the six participating countries.
* a numerical model has been used to estimate threshold exceedances.

Figure 2.